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The present study investigated the passive avoidance and spatial learning in the μ-opioid receptor gene knockout mice and wild type mice. In the step-through passive avoidance task, the μ-opioid receptor knockout mice did not differ from the wild type mice. In Morris water maze, however, the μ-opioid receptor knockout mice showed significant memory deficit compared to wild type mice. In the [^{3}H]pirenzepine autoradiographic binding for the muscarinic type 1 receptor, the [^{3}H]pirenzepine binding was selectively decreased in the dentate gyrus (10 %) of the hippocampus in μ-opioid receptor knockout mice compared to wild type. The acetylcholine level was reduced in the cortex of μ-opioid receptor knockout mice (22 %) compared to the control wild type mice. These results suggest that memory impairment in the μ-opioid receptor knockout mice may be related to the decrease of M1 receptor in dentate gyrus of the brain and reduction of acetylcholine level.
Therefore, these results suggest that lack of the μ-opioid receptor is accompanied with reduction of the cholinergic system, showing an impairment of spatial memory.

[PB3-8] [10/17/2002 (Thr) 13:30 - 16:30 / Hall C]

Microglial activation and tyrosine hydroxylase immunoreactivity in the substantia nigral region following transient focal ischemia in rats

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The temporal profiles of the changes of dopaminergic cell and microglial activation induced by transient cerebral ischemia was investigated in the substantia nigral region which lay outside ischemic areas of rat brain after middle cerebral artery occlusion (MCAO). Transient cerebral ischemia was induced by intraluminal occlusion of the right middle cerebral artery for 2 h and reperfusion was continued for 1, 2, 3, 7, 10, 14, 30, 60, and 120 days. Activated microglial cells were visualized with immunohistochemistry using OX-42 antibody. We also examined the ischemia-induced apoptotic cell death event in the substantia nigra (SN) at 1, 2, and 3 days. Activated microglial cells, as amoeboid morphology, visualized with OX-42 antibody were increased at 1 day and dramatically increased at 7 days postischemia. Activated microglia cells became reduced in the substantia nigra from 7 days later. At 2 and 4 months postischemia, the number of activated microglia cells were similar to those of 2 weeks after ischemia/reperfusion. These results suggest that microglial cells be rapidly activated and those activated forms be sustained at least for 1 week in the substantia nigra following transient focal cerebral ischemia induced by MCAO. The temporal profiles of the changes of dopaminergic cell identified with immunohistochemistry using tyrosine hydroxylase antibody are under study.

Poster Presentations - Field B4. Immunology

[PB4-1] [10/17/2002 (Thr) 13:30 - 16:30 / Hall C]

Immuno-modulator effect of Cefodizime in IL-6

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